

has an important influence. A deficient thyroid secretion causes the skin to feel dry, rough and scaly. In excessive deficiency, there is deposited in the skin a mucoid substance which does not pit as does oedema, and this gives a dull and stupid appearance to the face. The scalp and hair are dry and poorly nourished. The eyebrows are thin, especially at the outer ends. The nails are rough and brittle.

In excessive thyroid secretion, the skin is warm, moist and flushed, especially on the face and chest. Pressure leaves a pale place which remains colorless for several seconds. Dermographia is often present, and excessive sweating is common. Pigmented spots are often present on the hands, face and neck. In a general way the amount of thyroid secretion is correlated with the basal metabolism.

The functions of the pituitary gland are so far reaching, and so closely tied up with the other glands that it might well be the subject of a book, and only the most casual reference to its rôle as the leader of the band can be given at this time. Malfunction of the anterior pituitary teamed with the pars intermedia of the adrenal is associated with abnormal pigmentation, and sometimes with androgenic increase, which occasionally causes masculinization in women. Deficient secretion also may cause brown pigment, resembling freckles, and if there is a definite deficiency, as in Simmonds disease, there is a general atrophy of the skin and all other tissues, as in old age.

Numerous early investigators maintained that hormones have an important influence on the prevention of the ravages of age, and it was thought that rejuvenation could be brought about by increasing the supply of estrogenic or androgenic hormones; but this view has not been entirely upheld by subsequent investigators. It has some degree of truth, however, because sex hormones do retard some of the degenerations of the skin and, as a general rule, when there is a more youthful skin than the chronological age would warrant, the androgenic or estrogenic secretion is found to be higher than normal. There always exists a factor in the substratum which limits the extent of the hormone activity, and this is controlled by heredity. So, therefore, hormones can only accentuate or retard the natural tendencies of the individual.

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Wilms Tumor (Embryoma of the Kidney)

Max Wilms (1867-1918), while *Privat-dozent* in surgery at Leipzig under Trendelenburg, published in 1889 the first volume of a comprehensive trilogy "Die Mischgeschwülste [Mixed Tumors]" (Leipzig, 1902). The title of the first volume is "Die Mischgeschwülste der Niere [Mixed Tumors of the Kidney]." A portion of the translation from page 83 follows:

"I . . . am satisfied that as a result of the present treatise I have been able to widen our understanding of the structure and growth of mesodermal kidney tumors, and with the support of solid embryologic facts, to demonstrate their origin from definite cells derived from and corresponding in their characteristics to the mesoderm of the midportion of the body."—R. W. B., in *New England Journal of Medicine*.

SCRUB TYPHUS

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L. E., male, 30, an aviator, had been for 18 months in the South Pacific, visiting various islands outside the general area of known scrub typhus endemicity. He had been in poor health for some months.

REPORT OF CASE

Present Illness. The patient was hospitalized in the Pacific area for suspected cholecystitis with achylia. X-rays of the gastrointestinal tract and gallbladder were negative. Two weeks after entering the hospital, he developed a tender swelling above the right elbow in the region of the epitrochlear gland. He had had previous to this, for an uncertain time, a lesion on the terminal phalanx of the middle finger of the right hand. His temperature rose to 99°-100° F. daily. Four days after appearance of the enlarged gland above the right elbow, the corresponding axillary glands enlarged, becoming tender and accompanied by redness, and swelling around the epitrochlear gland, with red streaks extending below the elbow and slightly above the region of the inflamed gland. He had a sensation of tightness and aching in both groins and in the right testicle. He had never had malaria. A tentative diagnosis was made of filariasis, although no microfilaria could be demonstrated.

The patient arrived in San Francisco on May 13th, and on May 20th was hospitalized in Mary's Help Hospital.

Examination, on May 13th, was normal, except for the following points. The liver descended 2 finger-breadths below the costal edge, and there was tenderness in the gallbladder area. There was considerable enlargement of the right epitrochlear and axillary glands, with extreme tenderness and much overlying cellulitis, no lymphangitis, and moderate redness. There was a lesion on the inner side of terminal phalanx of right middle finger, about 8 mm. in diameter, raised, smooth except for some umbilication, red, not necrotic and with no surrounding inflammation. Temperature rose 1-2° F. in the afternoon and evening. The spleen was not palpable. Filariasis, malaria and cholecystitis were suspected.

Blood count, on May 13th was as follows:—Hgb. (Newcomer) 110 per cent, (18.6g.). Red cells, 5,630,000. Color index, 0.98. White cells, 12,200; polynuclear cells, 73 per cent, of which 63 per cent were mature and 10 per cent were stab cells. Lymphocytes, 18 per cent. Monocytes, 6 per cent. Eosinophiles, 2 per cent. Basophiles, 1 per cent. Kolmer and Kahn reactions were negative. On May 17th the white cells were 9,700 with 65 per cent polynuclear neutrophils of which: 11 per cent were stab cells, lymphocytes, 29 per cent, monocytes, 5 per cent and eosinophiles, 1 per cent. On May 19th, the leucocytes stood at 13,050 with 53 per cent polynuclear neutrophils (17 per cent stab cells), 37 per cent lymphocytes. Monocytes, 7 per cent, and eosinophiles, 3 per cent. Twice daily, from May 13th to May 20th, thick-and-thin blood films failed to show microfilaria, malarial plasmodia or other parasites. Icterus index was 10, Van Den Bergh reaction was negative, both direct and indirect. Stool specimens were free of parasites and the urine was normal.

The patient was given sulfadiazene 1.0 g. four times daily for 3 days, with no change in temperature or physical findings.

On May 20th, the patient was hospitalized. The preceding day a morbilliform uniform rash appeared over

the trunk and extended lightly to the arms and legs. The conjunctivae were deeply injected. The patient appeared acutely ill, apprehensive, alert and nervous. He felt seriously ill.

Examination showed right epitrochlear and axillary glands were enlarged. The small uniform macular rash did not extend to face, palms or soles, and did not become confluent or necrotic. The temperature course was irregular for the week before admission, ranging between 99° and 102.6°. In the second week it gradually began to remit and became normal late in the third week.

Blood count, on May 20 in hospital, was as follows:—Red cells, 5,330,000. Hgb., 100 per cent. White cells, 12,900, with polynuclear neutrophils, 85 per cent, lymphocytes, 10 per cent, monocytes, 4 per cent and eosinophils, 1 per cent. Subsequent blood counts were essentially similar. Icterus index was 10. Repeated examinations for malarial plasmodia were negative. No microfilaria were found, including study of thick films at 2-hour intervals, one night. Blood cultures on May 22, 23, and 24 were negative. Blood agglutinations were negative for tularemia, brucella, dysentery bacilli and the typhoids. Heterophile agglutination of sheep red cells by patient's serum was negative (1-56). Several agglutination tests of blood serum against fresh cultures of proteus OX 19 were negative. Agglutination tests with proteus OX 2 were negative. On May 29, fresh culture of proteus OX K gave a positive titer of 1:80 and, 2 days later, of 1:160. Four days after this, the titer was again 1:80.

A few drops of gland juice were obtained from the swollen epitrochlear gland. Cultures failed to show growth. Inoculation of three white mice gave no evidence of illness. The mice remained normal, until sacrificed on the 10th and 14th days after inoculation. Examination by Dr. J. J. Hawthorne failed to show rickettsial or bacterial infection.

COMMENT

Clinical Course. The rash began to recede after 2 days and disappeared in 6 days. The spleen became palpable on May 30th. The epitrochlear gland, after diagnostic puncture, suppurated, discharged for 10 days and gradually healed. Clinical improvement accompanied the lytic fall of temperature, and the patient left the hospital on May 31st. He was feeling normal except for considerable prostration. In the subsequent 2 months he regained his normal weight and strength.

Treatment was symptomatic, using aspirin, codein, cool sponging and, one period of 3 days, of sulfadiazene when secondary bacterial infection was feared. Epsom salt compresses and, later, alcohol-glycerin compresses were used on the suppurating gland. Special attention was given to a high caloric diet and excess vitamins in general. Sedatives were necessary for sleep.

Diagnosis. 1. Mild subacute cholecystitis.

2. Scrub typhus.

This diagnosis rested on:

(1) recent presence in an endemic area with fairly close contact with rats (hence the probability of transfer of larval Trombiculae from rat to patient);

(2) the clinical picture of primary lesion (but not necrosing), injected conjunctivae, severe subjective symptoms, fever ending by lysis, charac-

teristic rash, proximal lymphadenitis, and enlarged spleen;

(3) positive OX K agglutination with negative OX 19 and OX 2, and elimination of diagnoses of plague, tularemia, and malaria. Filariasis could not be excluded certainly as a concomitant diagnosis.

The atypical features were the absence of leucopenia, the nonnecrotic primary lesion, the apparently long incubation period (uncertain, probably three weeks or more), the absence of pulmonary involvement and deafness, and the suppuration of a gland (this may have been precipitated by diagnostic gland puncture). However, suppuration occasionally is seen in scrub typhus and increases the resemblance to plague bubo.

Conclusion. Imported cases of scrub typhus may be seen at any time, anywhere in the United States.

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WAR ANESTHESIA IN THE SOUTH PACIFIC*

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BEFORE presentation, it was necessary to have this paper censored by the Navy Department; consequently, statistics will be limited, as action is, and will be going on in the South Pacific for some time.

The purpose of the paper is to show you, if possible, how difficult and unsatisfactory anesthesia is away from the modern operating room, and to let any Service-connected individual in this audience know what to expect should he be sent into the war area. Several weeks have passed since these experiences, but anesthesia has not yet been, and cannot be altered to any extent.

Our organization was a so-called Mobile Base hospital—*mobile*, because we brought all of our equipment with us and set up the hospital ourselves; *base*, because, once set up, it cannot easily be moved. Each doctor was given a crew of men and set to work putting up the prefabricated steel buildings; our job was to lay down the wooden floors. When you consider that this hospital had 1,000 acute, and 2,500 ambulatory or convalescent beds, you have some idea of the amount of boards and nails we used. Long before the unit was completed, patients began arriving, so that it was often necessary to leave the construction and rush to the surgeries which were built first. We often worked by lantern light to complete a building in order to get more wounded men in out of the weather. In all the times it was necessary to awaken and bring out my crew, but not once did any fail to show up or grumble

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